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Enterovirus-infected immune cells of spleen and lymph nodes in the murine model of chronic myocarditis: a role in pathogenesis?

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Molecular hybridization studies have demonstrated that human enteroviruses, including group B coxsackieviruses (CVB), are detectable not only in endomyocardial biopsies of patients with acute enterovirus myocarditis but also in those with chronic disease. Such infections are observed in some patients with end-stage dilated cardiomyopathy, indicating the possibility of persistent heart muscle infection. Enterovirus persistence in the human heart is supported by the recent discovery in various murine models of enterovirus myocarditis that chronic inflamed heart muscle lesions are consistently associated with enterovirus persistence. Application of in-situ hybridization in a multiorgan study of CVB3-infected immunocompetent mice now reveals that, in addition to the myocardium, spleen and lymph nodes are persistently infected. During acute myocarditis, the majority of infected spleen cells was found to be located within the follicles of spleen and lymph nodes. At later stages of the disease, enteroviral infection was shown to be restricted to cells of the germinal centre in secondary follicles of spleen and lymph nodes. Thus, infected immunocompetent cells may play an important role in dissemination of the virus in the host and maintenance of a non-cardiac viral reservoir.

PMID: 8682099 [PubMed - indexed for MEDLINE]

1: Eur Heart J. 1995 Nov;16 Suppl K:42-8. Related Articles, Links

Role of angiotensin converting enzyme inhibitors in preventing left ventricular remodelling following myocardial infarction.

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Progressive changes typically occur in left ventricular (LV) architecture following moderate- to large-sized myocardial infarction (MI). These changes include early expansion and thinning of the infarct zone and subsequent increase in myocardial mass within the non-infarcted zone, with LV dilatation and loss of the normal elliptical configuration of the LV cavity. These changes are accompanied by impaired myocyte function and advancing clinical expression of heart failure. Numerous animal and human studies have documented inhibition of LV remodeling post-MI by angiotensin converting enzyme (ACE) inhibitors. Although the ideal timing for initiating treatment remains uncertain, evidence exists that benefit persists long after the time of initial injury. Mechanisms for the effects of ACE inhibitors on LV remodelling may be dependent on changes in myocardial load, may be load independent, or both. These effects are likely to be mediated by reductions in circulating and local tissue concentrations of angiotensin II and in bradykinin degradation. Regardless of the exact mechanism or mechanisms by which ACE inhibitors exert their favourable influence on LV remodelling, it is likely that this effect is a key mediator of the documented clinical benefits afforded by treatment with this class of agents.

Publication Types:
- Clinical Trial
- Review
- Review, Tutorial

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Quantitative aspects in myocardial contrast echocardiography.

Rovai D, Ferdeghini EM, Mazzarisi A, Paterni M, Lubrano V, Vassalle C, Serasini L, L'Abbate A.

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Myocardial tissue perfusion is not currently quantified in the clinical setting. Thus the aim of this paper is to review the quantitative information on myocardial perfusion provided by contrast echocardiography. In a circulatory model-without the capillary network interposed between injection and sampling point of contrast-the transit time of microbubbles (source of the echo contrast effect) is inversely related to absolute flow, thus providing accurate quantitation. A similar situation is represented by blood flow inside a vessel or a cardiac cavity, where, if the prerequisites for quantitation are respected, it is possible to measure blood flow by contrast echocardiography. In the coronary circulation, the transit time of contrast microbubbles varies according to their interaction with coronary microcirculation, and to the characteristics of contrast agents as flow tracers. Echo contrast agents with small microbubbles have been injected into the coronary branches of experimental animals, under both coronary autoregulation and maximal coronary dilation, providing good estimates of coronary blood flow. The accuracy of these measurements might improve when new contrast agents, with characteristics closer to those of a flow tracer, are available. If a tracer is injected before a bifurcation, and provided it mixes adequately, the amount of tracer distributed to each branch is proportional to the corresponding blood flow. A similar situation is encountered when an echo contrast agent is injected into the aortic root or into the left main coronary artery. Here, the ratio between myocardial signal intensity in the different perfusion territories reflects the corresponding ratio of blood flows. The validity of this approach has been previously demonstrated in experimental animals and validated in patients with coronary stenoses. The injection of contrast agents into the coronary circulation at baseline and under coronary hyperaemia has the potential for measuring coronary blood flow reserve. However, what is still unclear is whether contrast echo changes reflect changes in coronary blood flow (i.e. flow reserve), coronary blood volume (i.e. coronary recruitment) or both, and also whether they influence the different types of contrast agent. Finally, myocardial contrast echocardiography can provide information on the spatial distribution of myocardial perfusion, i.e. the presence, site and extent of perfused myocardium. Thus, in models where myocardial perfusion may be either present or absent, contrast echo can provide an accurate estimate of

Fibrinogen and cerebrovascular disease.

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The importance of fibrinogen has been identified in two prospective observational studies. Reactive elevations in fibrinogen levels that occur within hours of a major stroke invalidate most cross-sectional case-control studies evaluating fibrinogen as a risk factor. However, as no elevation is seen following fresh episodes of transient ischaemic attacks, reliable conclusions drawn from a case-control study using such patients support the findings of the prospective studies. The association is related to occlusive stroke, but the relationship with intracerebral haemorrhage is unclear. The relationship has been found to be independent of other haemostatic and haemorheological factors (e.g. von Willebrand factor, tissue plasminogen activator and packed cell volume). Adjustment for regression dilution bias would further strengthen the observed relationship. Therefore, after blood pressure, fibrinogen is the most important potentially treatable risk factor for ischaemic stroke. There are several mechanisms whereby fibrinogen could promote athero-thromboembolism: thrombosis through a hypercoagulable state; the acceleration of atherosclerosis; or the reduction of blood flow due to high blood or plasma viscosity. The mechanism, however, is unlikely to be mediated through high blood viscosity per se as secondary erythrocytosis (another major determinant of blood viscosity) has not consistently been found to be a risk factor for stroke. Studies relating fibrinogen levels to the degree of carotid artery stenosis support the accelerating influence of fibrinogen on atherosclerosis. Fibrinogen should be considered a risk factor for ischaemic stroke and included in the assessment of individual risk factors.(ABSTRACT TRUNCATED AT 250 WORDS)

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